Chapter 5

Air Toxics

Background

Hazardous air pollutants (HAPs), commonly referred to as air toxics or toxic air pollutants, are pollutants that cause, or may cause, adverse health effects or ecosystem damage. The CAA lists 188 pollutants or chemical groups as hazardous air pollutants in section 112 (b)(1) and targets sources emitting them for regulation.[1] Examples of air toxics include heavy metals like mercury and chromium; organic chemicals like benzene, 1,3-butadiene, perchloroethylene (PERC), dioxins, and polycyclic organic matter (POM); and pesticides such as chlordane and toxaphene.

HAPs are emitted from literally thousands of sources including stationary (large industrial facilities such as utilities and smaller, area sources like neighborhood dry cleaners) as well as mobile sources (automobiles). Adverse effects to human health and the environment due to HAPs can result from exposure to air toxics from individual facilities, exposure to mixtures of pollutants found in urban settings, or exposure to pollutants emitted from distant sources that are transported through the atmosphere over regional, national or even global air sheds. Exposures to HAPs can be either short-term or long-term in nature. In some cases, effects can be seen immediately, such as those rare instances in which there is a catastrophic release of a lethal pollutant, or when a respiratory irritant is regularly released in sufficient levels to cause immediate effects. In other cases, the resulting effects may be experienced from long-term exposure (e.g., from mercury), over a period of several months or years.

In addition to breathing air contaminated with air toxics, people can also be exposed to some HAPs through other, less direct pathways such as through the ingestion of food from contaminated waters. Some air toxics bio-accumulate in body tissues, resulting in predators building up large concentrations from consuming contaminated prey, thereby magnifying up the food chain (i.e., each level accumulates the toxics and passes the burden along to the next level of the food web.) Presently, over 2,100 U.S. water bodies are currently under fish consumption advisories, representing approximately 15 percent of the nation's total lake acreage, and 5 percent of the nation's river miles. In addition, the Great Lakes and a large portion of the U.S. coastal areas are also under fish consumption advisories. Mercury, polychlorinated biphenyls (PCBs), chlordane, dioxins, and dichlorodiphenyltrichloroethane (DDT) and its degradation products: dichlorodiphenyldichloroethylene (DDE) and dichlorodiphenyldichloroethane (DDD), were responsible for almost 95 percent of all fish consumption advisories in effect in 1996.[2]

Health and Ecological Effects

Compared to information for the criteria pollutants previously described in other chapters, the information concerning potential health effects of the HAPs (and their ambient concentrations) is relatively incomplete. Most of the information on potential health effects of these pollutants is derived from experimental animal data. Enough evidence exists, however, to conclude that air toxics may pose a risk of harmful effects to public health and the environment. Potential health effects resulting from exposure to HAPs include leukemia and other cancers; reproductive and developmental effects such as impaired development in newborns and young children, inability to complete a pregnancy and decreased fertility; and damage to the pulmonary system. Of the 188 HAPs referenced previously, almost 60 percent are classified by EPA as known, probable or possible carcinogens. Nearly 30 percent of the HAPs have some evidence of reproductive or developmental effects (mostly in experimental animal data); about 13 percent are suspected endocrine disruptors; and approximately 60 percent may effect the central nervous system (CNS) and/or create other adverse effects such as irritation of the lungs. The extent to which these effects actually occur in the population depends on a number of factors, includ-

ing the level and duration of the exposure to the pollutant(s).

Toxic air pollutants can have a number of environmental impacts in addition to the threats they pose to human health. Animals, like humans, may experience health problems if they breathe sufficient concentrations of HAPs over time. Little quantitative information currently exists, however, describing the nature and scope of the effects of air toxics on non-human species. One of the more documented ecological concerns associated with toxic air pollutants is the potential for some to damage aquatic ecosystems. In some cases, deposited air pollutants can be significant contributors to overall pollutant loadings entering water bodies. For the Great Lakes, international workshops have examined the importance of deposition of air toxics, relative to other loadings. While data are presently insufficient for quantitative estimates comparing air deposition and other loading pathways (especially for persistent chemicals which continue to move among air, water, and sediments), deposition of air toxics to the Great Lakes is considered potentially significant and continues to be investigated under a binational monitoring network.[3] A number of studies suggest that deposited air toxics contribute to deleterious effects such as birth defects, reproductive failures, developmental disorders, disease, and premature death in fish and wildlife species native to the Great Lakes. Persistent air toxics are of particular concern in these aquatic ecosystems, as levels bio-accumulate in animals at the top of the food chain resulting in exposure many times higher than that indicated from the water or air.

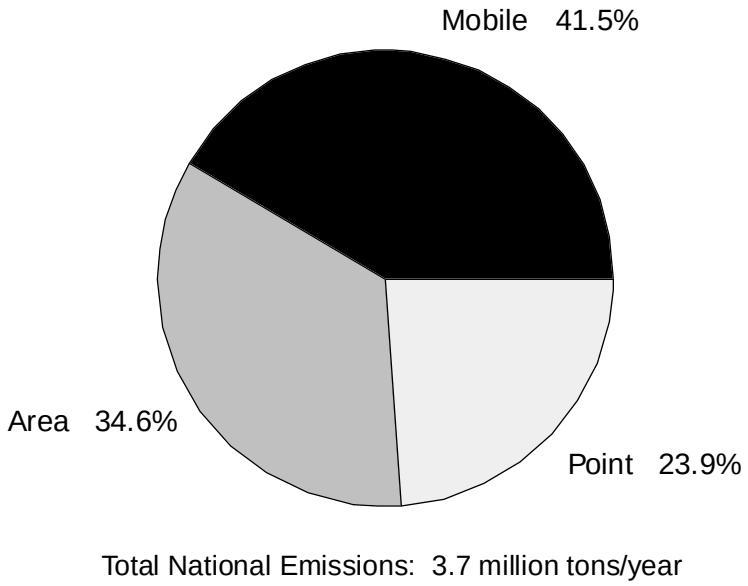

Figure 5-1. Total national HAP emissions by source type, 1993.

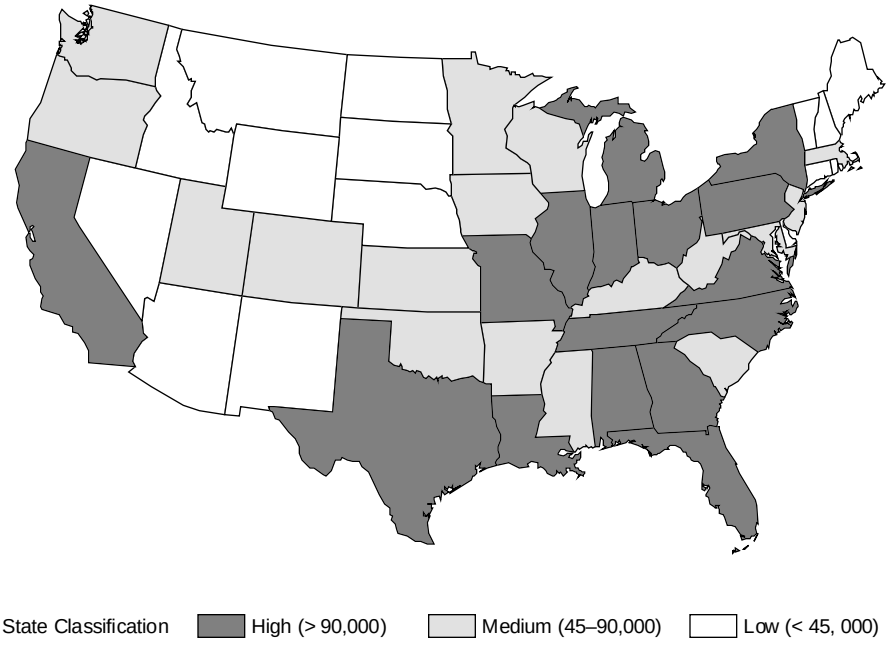

Figure 5-2. HAP emissions by state, 1993 (tons/year).

Table 5-1. Top 20 Sources of 1993 Toxic Emissions of Hazardous Air Pollutants

Rank	Source Category	Emissions(tpy)	Major HAPs by mass/category
1.	Mobile Sources: On-Road Vehicles	1,389,111	Acetaldehyde, Benzene, 1,3-Butadiene, Formaldehyde, Toluene, Xylenes
2.	Consumer & Commercial Product Solvent Use	414,096	Methanol, Methyl chloroform, Toluene, Xylenes
3.	Open burning: Forests and Wildfires	207,663	Acetaldehyde, Acrolein, Benzene, 1,3-Butadiene, Formaldehyde, Toluene, Xylenes[4]
4.	Glycol Dehydrators (Oil and Gas Production)	206,065	Benzene, Toluene, Xylenes
5.	Mobile Sources: Non-Road Vehicles & Equip.	145,866	Acetaldehyde, Benzene, 1,3-Butadiene, Formaldehyde
6.	Open Burning: Prescribed Burnings	134,149	Acetaldehyde, Acrolein, Benzene, Formaldehyde[4]
7.	Residential Boilers: Wood/Wood Residue	98,646	Acetaldehyde, Benzene, POM Combustion[5]
8.	Dry Cleaning: Perchloroethylene	95,700	Perchloroethylene
9.	Organic Chemical Manufacturing	91,419	Benzene, Ethylene glycol, Hydrogen chloride, Methanol, Methyl chloride, Toluene
10.	Pulp and Paper Production	88,579	Acetaldehyde, Benzene, Carbon tetrachloride, Formaldehyde, Hydrochloric acid, Methanol, Methylene chloride
11.	Halogenate Solvent Cleaning (Degreasing)	61,374	Methyl chloroform, Methylene chloride, Perchloroethylene, Trichloroethylene
12.	Primary Nonferrous Metals Production	37,980	Chlorine, Hydrogen chloride, Metals
13.	Cellulosic Man-Made Fibers	37,605	Carbon disulfide, Hydrogen chloride
14.	Petroleum Refining (All Processes)	27,115	Benzene, Hydrochloric acid, Toluene, Xylenes
15.	Municipal Waste Combustion	24,777	Formaldehyde, Hydrogen chloride, Manganese, Mercury, Lead
16.	Motor Vehicles (Surface Coating)	23,081	Methyl chloroform, Toluene, Xylenes
17.	Gasoline Distribution Stage II	21,512	Benzene, Glycol ethers, Naphthalene, Toluene
18.	Utility Boilers: Coal Combustion	21,404	Hydrogen fluoride, Manganese, Methylene chloride, Selenium[6]
19.	Plastics Materials and Resins Manufacturing	20,830	Methanol, Methylene chloride, Styrene, Vinyl acetate
20.	Flexible Polyurethane Foam Production	19,550	Methylene chloride

Emissions Data

There are approximately 3.7 million tons of air toxics released to the air each year according to OAQPS' NTI. Air toxics are emitted from all types of manmade sources, including large industrial sources, small stationary sources, and mobile sources. As shown in Figure 5-1, the NTI estimates of the area source (sources of HAPs emitting less than 10 tons per year of an individual HAP or 25 tons per year of aggregate emissions of HAPs each) and mobile source contributions to the national emissions of HAPs are approximately 35 and 41 percent respectively.

As part of the characterization of sources of HAPs nationwide, a listing of the sources emitting the greatest quantities of HAPs is presented in Table 5-1 for the 1993 inventory. These sources do not necessarily represent those which pose greatest risk. HAP emissions are not equivalent to risks posed by exposure to these compounds because some of the HAPs are more toxic than others, and actual exposures will vary by site-specific conditions such as stack height, topography, wind speed and direction, and receptor location. The data in Table 5-1, however, do provide an indication of the variety of sources and HAPs which are emitted from such sources in relatively large quantities.

Table 5-1 also shows the major contributing HAPs for each of the top 20 source categories. The 20 sources listed in Table 5-1 accounted for 87 percent of total emissions of the 188 HAPs for the year 1993. The first two source categories, on-road motor vehicles (a mobile source category) and consumer / commercial solvent use (an area source category) account for approximately 47 percent of the 188 HAPs emitted annually. Figure 5-2 is presented to illustrate the geographic distribution of emissions of HAPs by mass. This figure shows total emissions of HAPs for each state and does not necessarily imply relative health risk by exposure to HAPs by state. The categorization of pollutant emissions as high, medium, and low provides a rough sense of the distribution of emissions. In addition, some states may show relatively high emissions as a result of very large emissions from a few facilities or show relatively large emissions as a result from many very small point sources.

The NTI, which is currently being updated, includes emissions information for 188 HAPs from 913 point-,

area-, and mobile-source categories. TRI data were used as the foundation of this inventory. The TRI data, however, are significantly limited in several key aspects as a tool for comprehensively characterizing the scope of the air toxics issue. For example, TRI does not include estimates of air toxics emissions from mobile and area sources.[7] The NTI suggests that the TRI data alone represent less than half of the total emissions from the point source category. Therefore, the NTI has incorporated other data to create a more complete inventory.

Data from OAQPS studies, such as the Mercury Report,[8] and 112c(6) and 112(k) inventory reports, and data collected during development of Maximum Achievable Control Technology (MACT) Standards under section 112(d), supplement the TRI data in the NTI. In addition, state and local data such as the California Air Resource Board's (CARB) Hot Spots Inventory, Houston Inventory, and the Arizona HAP Study were incorporated in the 1993 NTI. The use of non-TRI data from other sources is particularly important for providing estimates of area- and mobile-source contributions to total HAP emissions. Note that development of the NTI is continuing and that additional information concerning emissions from sources regulated under the MACT program will be added, as well as additional state and local emissions data submitted as part of Title V operating permit surveys of the Act.

Table 5-2. Summary of Changes in Mean Concentration for HAPs Measured as a Part of the PAMS Program (24-hour measurements), 1994–1996*

	1994 to 1995			1995 to 1996		
HAP	# Sites	# Up	# Down	# Sites	# Up	# Down
Acetaldehyde	0	n/a	n/a	2	0	0
Benzene	7	0	4	5	1	2
Ethyl benzene	8	0	2	5	0	2
Formaldehyde	0	n/a	n/a	2	0	0
Hexane	5	2	0	4	0	0
Toluene	8	0	5	5	0	1
Styrene	7	0	1	5	1	2
m/p-Xylene	8	0	4	5	0	0
o-Xylene	7	0	1	5	0	1
2,2,4-Trimethylpentane	4	1	1	5	0	3

* Note that the terms "#Up" and "#Down" refer to the number of sites in which the change in annual mean concentration between 1994 and 1995, or 1995 and 1996, is a statistically significantly increase or decrease. The total number of sites (# sites) may not necessarily equal the sum of the corresponding "#Up" and "#Down" categories.

Table 5-3. Comparison of Loading Estimates for the Great Lakes

Chemical	Year	Superior (kg/yr)	Michigan (kg/yr)	Huron (kg/yr)	Erie (kg/yr)	Ontario (kg/yr)
PCBs (wet/dry)	1988	550	400	400	180	140
	1992	160	110	110	53	42
	1994	85	69	180	37	64
DDT (wet/dry)	1988	90	64	65	33	26
	1992	34	25	25	12	10
	1994	17	32	37	46	16
B(a)P	1988	69	180	180	81	62
	1992	120	84	84	39	31
	1994	200	250	na	240	120
Pb (wet/dry)	1988	230,000	540,000	400,000	230,000	220,000
	1992	67,000	26,000	10,000	97,000	48,000
	1994	51,000	72,000	100,000	65,000	45,000

Ambient Air Quality Data

Presently, there is no national ambient air quality monitoring network designed to perform routine measurements of air toxics levels. Therefore, ambient data for individual air toxic pollutants is limited (both spatially and temporally) in comparison to the data available from the long-term, nationwide monitoring for the six criteria pollutants. EPA has several efforts underway which, although less optimal than a comprehensive and routine HAPs network, will provide some information useful to assessing the toxics issue.

The Agency's PAMS collect data on concentrations of ozone and its precursors in 21 areas across the nation classified as serious, severe or extreme nonattainment areas for ozone. Because several ozone precursors are also air toxics, ambient data collected from PAMS sites can be used for limited evaluations of toxics problems in selected urban areas as well as assessment of the tropospheric ozone formation. Despite some limitations, the PAMS sites will provide consistent, long-term measurements of selected toxics in major metropolitan areas. The PAMS program requires routine measurement of 10 HAPs: acetaldehyde, benzene, ethyl benzene, formaldehyde,

hexane, styrene, toluene, m / p-xylene, o-xylene and 2,2,4-trimethlypentane.

Preliminary analysis of measurements of selected HAPs in PAMS areas indicate that concentrations of certain toxic VOCs in those areas appear to be declining. Table 5-2 shows 2-year comparisons for 24-hour measurements for nine air toxics measured at PAMS sites for the periods 1994–1995 and 1995–1996.[9] The only pollutant with more sites significantly increasing (at the 5-percent level) than those significantly decreasing (at the 5-percent level) for either time period, is hexane between 1994 and 1995. For a more detailed discussion of the PAMS program, see Chapter 4 of this report.

In addition to the PAMS program, EPA continues to administer and support voluntary programs through which states may collect ambient air quality measurements for suites of toxics. These programs include the Urban Air Toxics Monitoring Program (UATMP), as well as the Non-Methane Organic Compound (NMOC) and Speciated Non-Methane Organic Compound (SNMOC) monitoring programs. The UATMP is the "participatory" program dedicated to toxics monitoring which involves measurements of 37 VOCs and 13 carbonyl compounds.[10] In the current programs, five states are participating and operating 15 ambient measurement sites for toxics.[11]

Further, the Integrated Atmospheric Deposition Network (IADN), a joint U.S. / Canada measurement program, was initiated in 1990 to assess the relative importance of atmospheric deposition to the Great Lakes, and to provide information about sources of these pollutants.[12] The network consists of master (research-grade) stations on each lake, with additional satellite stations. There are two master stations in Canada and three in the United States that were chosen to be representative of regional deposition patterns. In addition to precipitation rates, temperature, relative humidity, wind speed and direction, and solar radiation collected at each site, concentrations of target chemicals are measured in rain and snow (wet deposition), airborne particles (dry deposition), and airborne organic vapors.[13]

The results of a comparison of deposition estimates from studies performed in 1988, 1992, and 1994 are presented in Table 5-3. Since the earlier estimates were based on sparse and uncertain data, these results are difficult to interpret definitively. The most consistent trend, however, is the reduction in 1994 lead deposition versus 1988 values for all the lakes, which is not surprising given the ban of leaded gas in the United States. Estimates of wet and dry deposition of PCBs to the lakes for 1994 show a decline compared to past estimates.[14] In addition, measurements of ambient air quality levels of PCBs at surface sites near Lake Superior appear to have remained constant over time compared to ambient levels near Lakes Erie and Michigan which have indeed declined. These downward trends in ambient air quality concentrations support estimations of an atmospheric half-life for PCBs of approximately six years which corresponds well to PCB half-lives seen in other environmental media.[15] The loading of one of the most toxic polynuclear aromatic hydrocarbons (PAH) yet characterized, benzo(a)pyrene (B(a)P), to the lakes seems to have increased; however, this is probably due to an underestimation of B(a)P in the 1992 studies.[16] Finally, the 1994 results show that DDT wet and dry deposition declined between 1988 and 1992, but rose slightly for all lakes except Superior in 1994.[17]

Concurrent with these monitoring efforts, EPA has recently initiated a program to identify, compile and catalogue all previously collected monitoring data for air toxics which is not now centrally archived. This effort is focusing presently on the compilation of measurements previously made by state and local agencies. These data will contribute to the development of an expanded and enhanced information infrastructure for air toxics.[18] All data completed as a result of this effort will be made universally accessible to all interested programs and analysts.

In addition, the Agency is also sponsoring a related project to develop environmental indicators based on air quality monitoring data, emissions data, modeling data, and administrative / programmatic data that can effectively demonstrate the extent and severity of the air toxics problem, and any progress made toward solving it in future years through regulatory or voluntary programs. Indicators will be included that consider population exposure and health risk, as well as ambient concentrations and emissions. Such indicators will be used to make geographic comparisons and assess temporal trends in subsequent trends reports.[19]

Air Toxics Control Program

The Regulatory Response

In 1990, Congress amended section 112 of the CAA by adding a new approach to the regulation of HAPs. This new approach first requires the development of technology-based emissions standards for the major sources of the 188 HAPs under section 112(d). The overall approach is to use available control technologies or changes in work practice to get emission reductions for as many of the listed HAPs as

possible, regardless of the HAP's inherent toxicity and potential risk. This technology-based standards program is commonly referred to as the MACT program. Although there is no health test in this phase, it is intended that effective MACT standards will reduce a majority of the HAP emissions and potential risks. Under Section 112(d)(6), the MACT standards are subject to periodic review and potential revision.

In addition, the CAAA calls for an evaluation of the health and environmental risks remaining after technology-based standards have been set (i.e., residual risks) and requires more stringent regulation if certain risk criteria are not met. Specifically, its focus is to achieve a level of protection that provides the public health with an "ample margin of safety" while also ensuring that residual emissions do not result in "adverse environmental effects."

Under the Urban Area Source Program, EPA is identifying at least 30 HAPs that are of particular concern when emitted in urban areas, especially from area sources. EPA currently is developing a plan to reduce emissions of such chemicals by regulating sources that account for 90 percent of the emissions and to reduce cancer incidence by 75 percent.

The CAAA also require EPA to conduct specific studies to evaluate other potential human health and ecological problems and to determine if regulation is necessary. The Agency is currently conducting studies of the atmospheric deposition to the Great Lakes and coastal waters,[20] the electric utility industry, and mercury. Updates for these studies are highlighted at the end of this chapter. EPA also is required under section 112(c)(6) of the CAA to identify sources of seven specific pollutants and to regulate sources accounting for 90 percent of the emissions of each.[21]

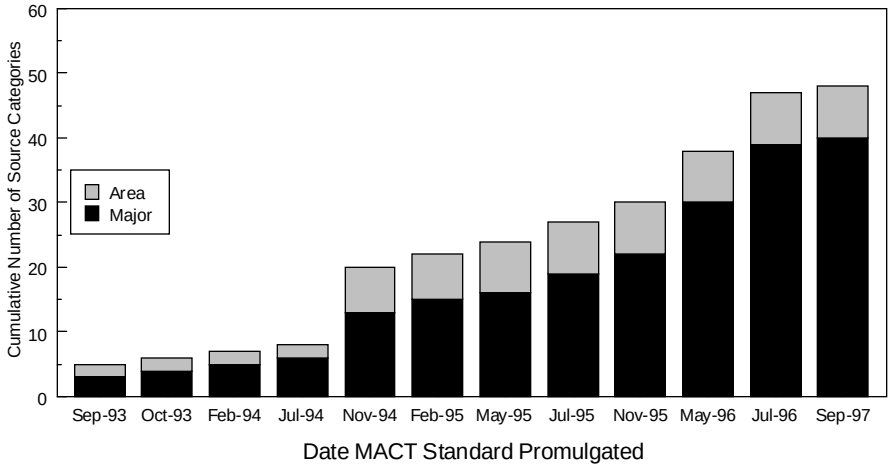

Figure 5-3. MACT source categories.

The air toxics program and the NAAQS program complement each other. Many air toxics are emitted in the form of particles or as VOCs which can be ozone precursors. Control efforts to meet the NAAQS for ozone and PM_{10} also reduce air toxic emissions. Furthermore, as air pollution control strategies for automobiles become more stringent, air toxic emissions from vehicles also are reduced. Requirements under the air toxics program can also significantly reduce emissions of some of the six NAAQS pollutants. For example, EPA's final air toxics rule for organic chemical manufacturing is expected to reduce VOC emissions by nearly 1 million tons annually.

The CAA recognizes that not all problems are national problems or have a single solution. National emission standards must be promulgated to decrease the emissions of as many HAPs as possible from major sources, but authority is also provided to look at smaller scale problems such as the urban environment or the deposition to water bodies in order to address specific concerns. The Act also recognizes the need to focus or rank efforts to meet specific needs, such as a concern for a class of toxic and persistent HAPs. There are mechanisms for increasing partnerships among EPA, states, and local programs in order to address problems specific to these regional and local environments.

Air Toxics Regulation and Implementation Status

The CAA greatly expanded the number of industries affected by national air toxic emissions controls. Large industrial complexes (major sources) such as chemical plants, oil refineries, marine tank vessel loading, aerospace manufacturers, steel mills, and a number of surface coating operations are some of the industries being controlled for toxic air pollution. Where warranted, smaller sources (area sources) of toxic air pollution such as dry cleaning operations, solvent cleaning, commercial sterilizers, secondary lead smelters, and chrome plating also are affected. EPA estimates that over the next 10

years the air toxics program will reduce emissions by 1.5 million tons per year.[22]

The emissions reductions are beginning to be realized for many industries. As many as 16 major- and eight area-source categories have begun to take some action toward complying with the controls required by the 2- and 4-year regulations. The extent of this compliance depends on the requirements of the regulations and actions taken by the industries to meet these requirements.

Emissions Reductions Through the MACT Program

The regulation of air toxics emissions through the process outlined in section 112 of the CAA, referred to as MACT regulations, is beginning to achieve significant emissions reductions of HAPs as well as criteria pollutants. As Figure 5-3 shows, as of September 1997 MACT standards have been promulgated for 48 source categories, representing all MACT standards in the 2- and 4-year groups plus one standard in the 7-year group. Sources are required to comply with these standards within three years of the effective date of the regulation, with some exceptions. Just recently to comply with section 112(s), EPA released a report to Congress describing the status of the HAP program under the CAA. EPA estimates that the 2- and 4-year standards will reduce HAP emissions by approximately 980,000 tons / year when fully implemented.[22] Concurrent control of particulate matter and VOC as ozone precursors by MACT standards, is estimated to reduce approximately 1,810,000 tons per year in combined emissions, a reduction that would not have occurred through other more conventional regulatory programs for these specific pollutants.

In addition, EPA has promulgated regulations on municipal waste combustors and hospital / medical / infectious waste incinerators under section 129 of the CAA which will significantly reduce emissions of the listed section 129 pollutants from these sources. These pollutants include particulate matter, sulfur dioxide, hydrogen chloride, oxides of nitrogen, carbon monoxide, lead, mercury, dioxins and dibenzofurans. For example, mercury emissions from municipal waste combustors are estimated to be reduced in the year 2000 by about 98 percent from 1990 levels. Mercury emissions from hospital / medical / infectious waste incinerators are estimated to be reduced by 93–95 percent, from 1995 levels, when the regulations become fully effective.

Residual Risk

To determine whether "post-MACT" risks are acceptable, Congress added a human health risk and adverse environmental effects-based "needs test" in the second regulatory phase. In this phase, referred to as "residual risk" standard setting, EPA is required to promulgate additional standards for those source categories that are emitting HAPs at levels that present an unacceptable risk to the public or the environment. Congress directed that such residual risk standards should "provide an ample margin of safety to protect public health." Non-cancer human health risks and adverse environmental effects will also be considered in setting residual risk standards. Using a risk management framework, EPA will determine whether technology-based emission standards sufficiently protect human health.

EPA is required by section 112(f)(1) of the Act to provide a report to Congress describing the methodology of approaches assessing these residual risks, the public health significance of any remaining risks, and technical and economic issues associated with controlling the risks. The report is currently scheduled for publication in 1999.

Special Studies/Programs

As mentioned previously, the CAA requires EPA to conduct special studies to assess the magnitude and effects of air toxics focusing on specific sources, receptors, and pollutants. Summaries of the main efforts follow.

The Great Waters Program

Section 112(m) of the CAA requires the Agency to study and report to Congress every two years on the extent of atmospheric deposition of HAPs and other pollutants to the Great Lakes, the Chesapeake Bay, Lake Champlain, and coastal waters, and the need for new regulations to protect these water bodies. The pollutants of concern to this effort include nitrogen compounds, mercury, and pesticides in addition to other persistent, bioaccumulating HAPs. This program coordinates with extensive research programs to provide new understanding of the complicated issue of atmospheric deposition of air pollution to water bodies. New scientific findings will be incorporated into each required biennial report to Congress and appropriate regulatory recommendations will be made based on those findings. This statute provides the authority to introduce new regulations or influence those under development in order to prevent adverse effects from these pollutants to human health and the environment.

The Mercury Study

The Mercury Study is a comprehensive study of mercury emissions from an-

thropogenic sources in the United States, an assessment of the public health and ecological effects of such emissions, an analysis of technologies to control mercury emissions, and the costs of such control. The study is mandated by section 112(n)(1)(B) of the CAA because mercury is, as an element, eternally persistent as well as being bioaccumulative and the cause of fish consumption advisories in more than 39 states. A number of observations can be made regarding trends in mercury use and emissions. The overall use of mercury by industrial and manufacturing source categories has significantly declined. Industrial use of mercury declined by nearly 75 percent between 1988 and 1995. Much of this decline can be attributed to the elimination of mercury as a paint additive and the phase-out of mercury in household batteries. Reducing mercury in manufactured products is important because emissions of mercury are most likely to occur when these products are broken or discarded. Based on trends in mercury use, EPA predicts that manufacturing use of mercury will continue to decline. Chlorine production from mercury cell chlor-alkali plants will continue to account for most of the use in, and emissions from, the manufacturing sector. This industry has pledged, however, to voluntarily reduce mercury use by 50 percent by 2006. Secondary production of mercury may increase as more recycling facilities begin operations to recover mercury from discarded products and wastes. A significant decrease will occur in mercury emissions from municipal waste combustors and medical waste incinerators when the final regulations promulgated by EPA for these source categories are fully implemented. Emissions from both categories will decline by at least 90 percent

Table 5-4. List of Potential 112(k) HAPs

CAS Number	Name
79345	1,1,2,2-Tetrachloroethane
140885	Ethyl acrylate
79005	1,1,2-trichloroethane
106934	Ethylene dibromide (dibromoethane)
78875	1,2-Dichloropropane (propylene dichloride)
75218	Ethylene oxide
106990	1,3-Butadiene
107062	Ethylene dichloride (1,2-dichloroethane)
542756	1,3-Dichloropropene
50000	Formaldehyde
106467	1,4-dichlorobenzene
302012	Hydrazine
75070	Acetaldehyde
	Lead compounds
107028	Acrolein
	Manganese compounds
79061	Acrylamide
	Mercury compounds
107131	Acrylonitrile
74873	Methyl chloride (chloromethane)
	Arsenic compounds
75092	Methylene chloride (dichloromethane)
71432	Benzene
101688	Methylene diphenyl diisocyanate (MDI)
	Beryllium compounds
	Nickel compounds
117817	Bis(2-ethylhexyl)phthalate (DEHP) Polycyclic organic matter
	Cadmium compounds
91225	Quinoline
56235	Carbon tetrachloride
100425	Styrene
67663	Chloroform
127184	Tetrachloroethylene (perchloroethylene)
	Chromium compounds
79016	Trichloroethylene
	Coke oven emissions
75014	Vinyl chloride
	Dioxins/furans
75354	Vinylidene chloride (1,1-Dichloroethylene)

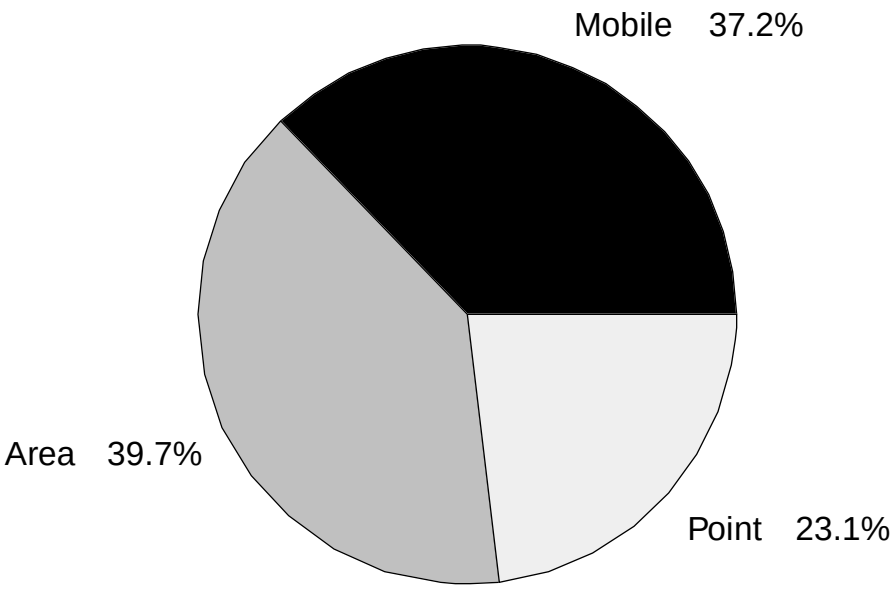

Figure 5-4. Emissions of 40 potential section 112(k) HAPs by source type (tons/year).

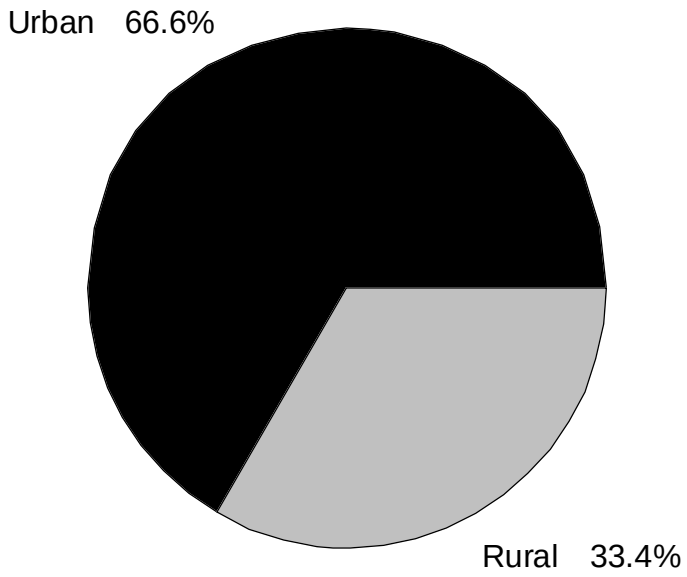

Figure 5-5. Emissions of 40 potential section 112(k) HAPs by urban and rural classification (tons/year).

from 1995 levels; to roughly 6 tons per year from municipal waste combustors and 1 ton per year from medical waste incinerators. In addition, EPA has proposed mercury emission limits for hazardous waste combustors. Based on 1995 estimates, coal-fired utility boilers are the largest remaining source category at 52 tons per year. Future mercury emissions from utility boilers depend on a number of factors including the nation's energy needs, fuel choices, industry restructuring and other requirements under the CAA (e.g., the Acid Rain Program). A recent EPA analysis also predicted mercury emissions will decline at least 11 tons per year as a result of implementation of the ambient standards for fine particulate matter. International efforts to reduce greenhouse gases will also reduce mercury emissions. The Mercury Study Report to Congress was completed in December 1997.

The Specific Pollutants Strategy

Section 112(c)(6) of the CAA requires EPA to identify sources of alkylated lead compounds, POM, mercury, hexachlorobenzene, PCBs, 2,3,7,8-tetrachlorodibenzo-p-dioxin, and 2,3,7,8-tetrachlorodibenzofuran, and then to subject sources accounting for not less than 90 percent of the aggregate emissions of each pollutant to standards.[22] Standards must be developed by EPA for sources of these HAPs that are not subject to current standards. In order to meet the requirements of section 112(c)(6), EPA compiled national inventories of sources and emissions of each of the seven HAPs.[23]

The Urban Area Source Program

Sections 112(c)(3) and 112(k) of the CAA require EPA to identify categories and subcategories of area sources of HAPs in urban areas that pose a threat to human health. Specifically, EPA must identify at least 30 HAPs that present the greatest threat to urban populations, and assure that sources accounting for 90 percent or more of the aggregate emissions of these 30 HAPs are subject to regulation. In addition, a national strategy must be developed to reduce cancer incidence attributable to these pollutants by at least 75 percent. In order to address the requirements of sections 112(c)(3) and 112(k), EPA compiled draft air emissions inventories of 40 potential urban HAPs, as seen in Table 5-4.[24]

Figures 5-4 and 5-5 present summary data from the draft urban air emissions inventory. Figure 5-4 indicates that: area sources account for 40 percent of emissions of the 40 potential urban HAPs, mobile sources account for 37 percent, and point (major) sources account for 23 percent. Figure 5-5 shows that urban emissions of the 40 potential HAPs account for 67 percent, and rural emissions account for 33 percent of the 40 potential HAPs.

It is important to note that emissions estimates do not necessarily reflect po-

tential health risk from exposure to these HAPs. Further analyses will be performed in conjunction with the development of the urban air toxics strategy. The development of the inventories for the potential urban pollutants, however, is a critical element in the regulatory strategy to reduce emissions of HAPs from area sources in urban geographic areas.

The Utility Air Toxics Study

As mandated by section 112(n)(1)(A) of the CAA, the Agency is studying HAP emissions from fossil fuel-fired (coal, oil, and gas) electric utilities and the associated hazards to public health. A draft utility report identifies 67 HAPs in the emissions database. The report predicts that over the next two decades there will be roughly a 30-percent increase in HAP emissions from coal-fired utilities and roughly a 50-percent decline in HAP emissions from oil-fired utilities. These projections are primarily based on anticipated energy demands and changes in fuel usage but also account for other factors such as expected controls.

References

1. This list originally included 189 chemicals. The CAA allows EPA to modify this list if new scientific information becomes available that indicates a change should be made. Using this authority, the Agency modified the list to remove caprolactam in 1996, reducing the list to 188 pollutants (*Hazardous Air Pollutant List;* Modification, 61 FR 30816, June 18, 1996).

2. "Update: Listing of Fish and Wildlife Advisories," announcing the availability of the 1996 update for the database: Listing of Fish and Wildlife Advisories (LFWA); U.S. EPA Fact Sheet, EPA-823-97-007, June 1997.

3. Hillery, B.R., Hoff, R.M., and Hites, R.A. 1997. "Atmospheric contaminant deposition to the Great Lakes determined from the Integrated Atmospheric Deposition Network." Chapter 15 in *Atmospheric Deposition of Contaminants to the Great Lakes and Coastal Waters*. 1997, Joel E. Baker, Editor. SETAC Press. (Society of Environmental Toxicology and Chemistry.)

4. POM is also a constituent of emissions of this source category, although not a major contributor to emissions on a mass basis.

5. One of the HAPs that is emitted from residential wood combustion is POM, which is a class of hundreds of compounds of varying toxicity. POM is defined in the NTI as the sum of 16 PAH compounds to provide a workable definition of the more toxic components of the class.

6. Mercury and hydrochloric acid are also constituents of emissions of this source category, although not major contributors to emissions on a mass basis.

7. In addition to the absence of emissions estimates for area and mobile source categories, there are other significant limitations in the TRI's portrayal of overall HAP emissions. First, facilities with Standard Industrial Classification (SIC) codes outside the range of 20 to 39 (the manufacturing SICs) are not required to report. Therefore, HAP emissions from facilities such as mining operations, electric utilities, and oil and gas production operations are not represented in the TRI. Further, TRI data are self-reported by the emitting facilities, and TRI does not require facilities to perform any actual monitoring or testing to develop their reported estimates. Consequently, the accuracy of the reported data may vary from facility to facility and from year to year. Finally, the original TRI list only required reporting for 173 of the 188 HAPs identified in the CAA.

8. Mercury Report to Congress, SAB review Draft. Volume II. An Inventory of Anthropogenic Mercury Emissions in the United States. EPA-452/R-96-001b.

9. Summaries of the health effects associated with the compounds included in this analysis are provided below:

   Acetaldehyde: The primary effects on humans, reported from short-term exposure to low to moderate levels of acetaldehyde, are irritation of eyes, skin, and respiratory tract. Short-term exposure effects on animals also include slowed respiration and elevated blood pressure. Effects on humans from long-term acetaldehyde exposure resemble those of alcoholism. Long-term exposures of animals have resulted in changes in respiratory tract tissues, as well as growth retardation, anemia, and kidney effects. While no information is available on acetaldehyde effects on human reproduction or development, both such effects have been observed in animal tests. Based on evidence of tumors in animals, EPA has classified acetaldehyde as a probable human carcinogen of relatively low carcinogenic hazard.

   Benzene: Reported effects on humans, from short-term exposure to low to moderate benzene levels, include drowsiness, dizziness, headache, and unconsciousness as well as eye, skin and respiratory tract irritation. Effects on both humans and animals from long-term benzene exposure include blood and immune system disorders. Reproductive effects have been reported for women exposed to high benzene levels and adverse effects on the developing fetus have been observed in animal tests. Changes in human chromosome number and structure have been reported under certain exposures. EPA has classified benzene as a known human carcinogen of medium carcinogenic hazard.

   Formaldehyde: Reported effects on humans, from short-term and long-term exposure to formaldehyde, are mainly irritation of eyes, nose, throat, and, at higher levels, the respiratory tract. Long-term exposures of animals have also resulted in damage to respiratory tract tissues. Although

little information is available on developmental effects to humans, animal tests do not indicate effects on fetal development. EPA has classified formaldehyde as a probable human carcinogen of medium carcinogenic hazard based on sufficient animal and limited human evidence.

Toluene: Effects on the CNS of humans and animals have been reported, from short-term exposure to low to moderate levels of toluene, and include dysfunction, fatigue, sleepiness, headaches, and nausea. Short-term exposure effects also include cardiovascular symptoms in humans and depression of the immune system in animals. CNS effects are also observed in long-term exposures of humans and animals. Additional long-term exposure effects include irritation of eyes, throat and respiratory tract in humans and changes in respiratory tract tissue of animals. Repeated toluene exposure has been observed to adversely affect the developing fetus in humans and animals. Due to a lack of information for humans and inadequate animal evidence, EPA does not consider toluene classifiable as to human carcinogenicity.

Xylenes: Reported effects on humans, from short-term exposure to high levels of xylenes, include irritation of eyes, nose, and throat, difficulty breathing, impairment of the CNS and gastrointestinal effects. Similar effects have been reported in animals in addition to effects on the kidney. Human effects from long-term exposure to xylenes are to the CNS, respiratory and cardiovascular systems, blood, and kidney. Long-term animal exposures to high levels of xylenes have shown effects on the liver. Effects on the developing fetus have been observed in animal studies. Due to a lack of information for humans and inadequate animal evidence, EPA does not consider xylenes classifiable as to human carcinogenicity.

Ethyl benzene: Effects reported, from short-term exposures of humans to high levels of ethyl benzene, include dizziness, depression of the CNS, eye, mucous membrane, nose and respiratory tract irritation, and difficulty breathing. In short-term exposures of laboratory animals, additional effects on the liver, kidney and pulmonary system have also been reported. Long-term exposures of animals have demonstrated effects on blood cells, the liver and kidneys. Effects on fetal development have also been observed in animal exposures. Due to a lack of information for humans and inadequate animal evidence, EPA does not consider ethyl benzene classifiable as to human carcinogenicity.

Styrene: Exposure to styrene vapors can cause irritation of eyes, nose, throat and respiratory tract in humans. Effects on the CNS of humans including dizziness, fatigue, sleepiness, headaches, nausea, and effects on intellectual function and memory have also been reported from long-term exposure to styrene. Long-term exposures of animals have demonstrated effects on the CNS, liver and kidney as well as eye and nasal irritation. Although the available information for humans is inconclusive, animal tests do not indicate effects on reproduction or fetal development. The carcinogenicity of styrene is currently under review by EPA. When absorbed into the human body, styrene is metabolized into styrene oxide, a direct acting mutagen that causes cancer in test animals.

Hexane: Reported effects on humans, from short-term exposure to high levels of hexane, include irritation of eyes, mucous membranes, throat and skin, as well as impairment of the CNS including dizziness, giddiness, headaches, and slight nausea. Long-term human exposure from inhalation is associated with a slowing of peripheral nerve signal conduction which causes numbness in the extremities and muscular weakness, as well as changes to the retina which causes blurred vision. Animal exposures to hexane have resulted in damage to nasal, respiratory tract, lung and peripheral nerve tissues, as well as effects on the CNS. No information is available on hexane effects on human reproduction or development. Limited laboratory animal data indicate a potential for testicular damage in adults, while several animal studies show no effect on fetal development. Due to a lack of information for humans and inadequate animal evidence, EPA does not consider hexane classifiable as to human carcinogenicity.

2,2,4-Trimethylpentane: Little information is available on the effects of 2,2,4-trimethylpentane overexposure in humans. Laboratory animals exposed to high levels for short periods have developed irritation, fluid build-up and bleeding in the lungs, as well as depression of CNS function. Kidney and liver effects have been reported from long-term animal exposures. No information is available on the potential for reproductive or developmental effects or on the carcinogenic potential of 2,2,4-trimethylpentane.

10. Twenty-eight of the 37 VOCs, and four of the 13 carbonyls measured as a part of the UATMP are defined as HAPs in section 112(b)(1) of the CAA.

11. The following states are presently participating in the UATMP: Arkansas, Louisiana, New Jersey, Texas, and Vermont.

12. The IADN fulfills legislative mandates in Canada and the United States that address the monitoring of air toxics. An international Great Lakes deposition network is mandated by Annex 15 of the *Great Lakes Water Quality Agreement between the United States and Canada*. In the United States, the CAA requires a Great Lakes deposition network.

13. The target chemicals include PCBs, pesticides, PAHs and metals. The compounds included as "target chemicals" were selected based on the following criteria: presence on List 1 of Annex 1 of the Great Lakes Water Quality Agreement (substances believed to be toxic and present in the Great Lakes); established or perceived water quality problem; presence on the International Joint Commission's Water Quality Board's list of criteria pollutants; evidence of presence in the atmosphere and an important deposition pathway; and feasibility of measurement in a routine monitoring network.

14. Hornbuckle, K.C., Jeremaison, J.D., Sweet, C.W., Eisenreich, S., "Seasonal Variations in Air-Water Exchange

of Polychlorinated Biphenyls in Lake Superior", J. Environ. Sci. Technol. 1994, 28, 1491-1501.

15. Hillery, B.R., Basu I., Sweet, C.W., Hites, R.A., *Temporal and Spatial Trends in a Long-Term Study of Gas-Phase PCB Concentrations near the Great Lakes*, Environ. Sci. Technol. 1997, 31, 1811-1816.

16. Hoff, R.M., Strachan, W.M.J., Sweet, C.W., D.F. Gatz, Harlin, K., Shackleton, M., Cussion, S., Chan, C.H., Brice, K.A., Shroeder, W.H., Bidleman, T.F., Atmospheric Deposition of Toxic Chemicals to the Great Lakes: A Review of Data Through 1994, Atmos. Environ., 1996, 30, 3505-3527.

17. Hillery, B.R., Hoff, R.M., Hites, R. Atmospheric Contaminant Deposition to the Great Lakes Determined from the International Atmospheric Deposition Network, In Atmospheric Deposition of Contaminants to the Great Lakes and Coastal Water, Baker, J.E., ed., Society for Environmental Toxicology and Chemistry, 1997.

18. Interest in participation in this voluntary effort and / or requests for further information about this data cataloguing effort should be directed to James Hemby, Office of Air Quality Planning and Standards, Mail Drop 14, Research Triangle Park, North Carolina 27711; 919-541-5459; and hemby.james@epamail.epa.gov.

19. The scheduled completion date for this project is September 1998; however, interim products will be released as completed. Additional information on this project is also available through James Hemby. Please see address and phone number above.

20. Section 112 (m) is commonly referred to as the "Great Waters" program.

21. These compounds, known as the section 112(c)(6) specific pollutants, are alkylated lead compounds, polycyclic organic matter, hexachlorobenzene, mercury, polychlorinated biphenyls, 2,3,7,8-tetrachlorodibenzofurans, and 2,3,7,8-tetrachlorodibenzo-p-dioxin.

22. Second Report to Congress on the Status of the Hazardous Air Pollutant Program Under the CAA, Draft. EPA-453 / R-96-015. October 1997.

23. The final inventory report is available at the following Internet address: www.epa.gov / ttn / uatw / 112cfac.html.

24. The draft inventory report is available at the following Internet address: www.epa.gov / ttn / uatw / 112kfac.html.